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Meeting abstract

Experiments to localize the site for the anxiogenic action of NPY mediated by Y_2 receptors in the mouse brain

Ramon O Tasan[1], Stefan Weger[2], Regine Heilbronn[2], Ngoc K Nguyen[3], Nicolas Singewald[3], Herbert Herzog[4] and Günther Sperk*[1]

Address: [1]Department of Pharmacology, Medical University of Innsbruck, Austria, [2]Institute of Virology, Charité, Free University of Berlin, Germany, [3]Department of Pharmacology and Toxicology, University of Innsbruck, Austria and [4]Garvan Institute of Medical Research, Darlinghurst Sydney, Australia

Email: Günther Sperk* - guenther.sperk@i-med.ac.at

* Corresponding author

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Neuropeptide Y (NPY) is abundant in the nervous system. It acts through Y_1, Y_2, Y_4 and Y_5 receptors and is involved in a variety of brain functions. When applied locally into the amygdala, NPY exerts an anxiolytic action, presumably mediated by Y_1 receptors. Depletion of Y_2 receptors induces an anxiolytic phenotype, possibly by abolishing the release-inhibiting action of presynaptic Y_2 receptors. In the present study we aimed to find the exact site of the presumed anxiogenic action mediated by Y_2 receptors. We conducted site-specific deletions of Y_2 receptors in $Y_2^{lox/lox}$ mice by local injection of an AAV-Cre vector into the hippocampus and the amygdala. As controls, an AAV-GFP vector was injected in $Y_2^{lox/lox}$ littermates at the same sites. Expression of Cre and GFP was verified by in situ hybridization and immunohistochemistry. Deletion of Y_2 receptors was visualized by receptor autoradiography and in situ hybridization. After bilateral injection of an AAV-Cre vector into the basolateral amygdala, mice revealed a tendency towards an anxiolytic phenotype in the light-dark test (LDT). When deletion of Y_2 receptors was confined to the central nucleus of the amygdala, an anxiolytic phenotype was observed in the elevated plus maze and the LDT. Moreover, a better stress coping ability was demonstrated in the tail suspension test. In contrast, no anxiolytic effect was detected after intrahippocampal injections. The experiments indicate that the anxiolytic and antidepressant-like effects of Y_2 receptor deletion may be generated in certain subnuclei of the amygdala.

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